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=> file biosis, medline, scisearch, embase
COST IN U.S. DOLLARS                          SINCE FILE      TOTAL
                                                   ENTRY    SESSION
FULL ESTIMATED COST                                0.21       0.21

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=> s sildenafil and zaprinast
L1          324 SILDENAFIL AND ZAPRINAST

=> display l1 all 324

L1   ANSWER 324 OF 324  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
     reserved on STN
AN   96234676  EMBASE   <<LOGINID::20070328>>
DN   1996234676
TI   Sildenafil (Viagra(TM)), a potent and selective inhibitor of
     type 5 CGMP phosphodiesterase with utility for the treatment of male
     erectile dysfunction.
AU   Terrett N.K.; Bell A.S.; Brown D.; Ellis P.
CS   Department of Discovery Chemistry, Pzifer Central Research,Sanwich, Kent
     CT13 9NJ, United Kingdom
SO   Bioorganic and Medicinal Chemistry Letters, (1996) Vol. 6, No. 15, pp.
     1819-1824. .
     ISSN: 0960-894X  CODEN: BMCLE8
CY   United Kingdom
DT   Journal; Article
FS   028     Urology and Nephrology
     029     Clinical Biochemistry
     030     Pharmacology
     037     Drug Literature Index
LA   English
SL   English
ED   Entered STN: 17 Sep 1996
     Last Updated on STN: 17 Sep 1996
AB   5-(2'-Alkoxyphenyl)pyrazolo[4,3-d]pyrimidin-7-ones, and in particular our
     preferred compound, sildenafil (VIAGRA(TM)), discovered through
     a rational drug design programme, are potent and selective inhibitors of
     the type 5 cGMP phosphodiesterase from both rabbit platelets and human
     corpus cavernosum.  Sildenafil is currently in the clinic for
     the oral treatment of male erectile dysfunction.
CT   Medical Descriptors:
     *corpus cavernosum
     *enzyme inhibition
     *penis erection
     *sexual dysfunction
     animal cell
     article
     controlled study
     drug design
     human
     human tissue
     nonhuman
     rabbit
```

structure activity relation
thrombocyte
Drug Descriptors:
*cyclic gmp phosphodiesterase: EC, endogenous compound
*phosphodiesterase inhibitor: CM, drug comparison
*phosphodiesterase inhibitor: AN, drug analysis
*phosphodiesterase inhibitor: DV, drug development
*sildenafil: CM, drug comparison
*sildenafil: AN, drug analysis
*sildenafil: DV, drug development
zaprinast: CM, drug comparison
RN (cyclic gmp phosphodiesterase) 9068-52-4; (sildenafil) 139755-83-2; (zaprinast) 37762-06-4
CN Viagra

=> FIL STNGUIDE

| COST IN U.S. DOLLARS | SINCE FILE ENTRY | TOTAL SESSION |
|---|---|---|
| FULL ESTIMATED COST | 8.70 | 8.91 |

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FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Mar 23, 2007 (20070323/UP).

=> display l1 all 323
YOU HAVE REQUESTED DATA FROM FILE 'BIOSIS, MEDLINE, SCISEARCH, EMBASE' - CONTINUE? (Y)/N:y

L1 ANSWER 323 OF 324 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 97375812 EMBASE <<LOGINID::20070328>>
DN 1997375812
TI The role of phosphodiesterase inhibitors in impotence.
AU Eardley I.
CS I. Eardley, Pyrah Department of Urology, St James' University Hospital, Leeds, United Kingdom
SO Expert Opinion on Investigational Drugs, (1997) Vol. 6, No. 12, pp. 1803-1810. .
Refs: 39
ISSN: 1354-3784 CODEN: EOIDER
CY United Kingdom
DT Journal; General Review
FS 028 Urology and Nephrology
030 Pharmacology
037 Drug Literature Index
LA English
SL English
ED Entered STN: 30 Dec 1997
Last Updated on STN: 30 Dec 1997
AB Penile erection is mediated by nitric oxide (NO) and its second messenger cyclic guanosine monophosphate (cGMP). The activity of cGMP is modulated by the enzyme phosphodiesterase (PDE). The most important physiological isoform of the enzyme in the penis is PDES. Sildenafil is a specific inhibitor of this enzyme which potentiates nitrergic cavernosal relaxation in vitro and in animals. Clinical studies suggest that it is effective in the treatment of male erectile dysfunction (MED), which would make it the first orally active therapy for this condition.

```
CT   Medical Descriptors:
     *impotence: DT, drug therapy
     human
     male
     oral drug administration
     pharmacodynamics
     review
     Drug Descriptors:
     *phosphodiesterase inhibitor: DV, drug development
     *phosphodiesterase inhibitor: DT, drug therapy
     *phosphodiesterase inhibitor: PK, pharmacokinetics
     *phosphodiesterase inhibitor: PD, pharmacology
       *sildenafil: DV, drug development
       *sildenafil: DT, drug therapy
       *sildenafil: PD, pharmacology
     cyclic gmp: EC, endogenous compound
     dipyridamole: PD, pharmacology
     phosphodiesterase: EC, endogenous compound
       zaprinast: PD, pharmacology
RN   (sildenafil) 139755-83-2; (cyclic gmp) 7665-99-8; (dipyridamole)
     58-32-2; (zaprinast) 37762-06-4


=> s "calcium blocker" or alpha-blocker or beta-blocker or "ergot alkanoid" or
"vasoactive intestinal peptide" or "dopamine agonist" or "opioid antagonist"
prostaglandin or "endothelin antagonist" or "potassium channel activator"
             0 "CALCIUM"
             0 "BLOCKER"
             0 "CALCIUM BLOCKER"
                 ("CALCIUM"(W)"BLOCKER")
             0 ALPHA
             0 BLOCKER
             0 ALPHA-BLOCKER
                 (ALPHA(W)BLOCKER)
             0 BETA
             0 BLOCKER
             0 BETA-BLOCKER
                 (BETA(W)BLOCKER)
             0 "ERGOT"
             0 "ALKANOID"
             0 "ERGOT ALKANOID"
                 ("ERGOT"(W)"ALKANOID")
             0 "VASOACTIVE"
             0 "INTESTINAL"
             1 "PEPTIDE"
             0 "VASOACTIVE INTESTINAL PEPTIDE"
                 ("VASOACTIVE"(W)"INTESTINAL"(W)"PEPTIDE")
             0 "DOPAMINE"
             0 "AGONIST"
             0 "DOPAMINE AGONIST"
                 ("DOPAMINE"(W)"AGONIST")
             0 "OPIOID"
             0 "ANTAGONIST"
             0 PROSTAGLANDIN
             0 "OPIOID ANTAGONIST" PROSTAGLANDIN
                 ("OPIOID"(W)"ANTAGONIST"(W)PROSTAGLANDIN)
             0 "ENDOTHELIN"
             0 "ANTAGONIST"
             0 "ENDOTHELIN ANTAGONIST"
                 ("ENDOTHELIN"(W)"ANTAGONIST")
             0 "POTASSIUM"
             0 "CHANNEL"
             0 "ACTIVATOR"
             0 "POTASSIUM CHANNEL ACTIVATOR"
```

```
                ("POTASSIUM"(W)"CHANNEL"(W)"ACTIVATOR")
L2            0 "CALCIUM BLOCKER" OR ALPHA-BLOCKER OR BETA-BLOCKER OR "ERGOT
                ALKANOID" OR "VASOACTIVE INTESTINAL PEPTIDE" OR "DOPAMINE AGONIS
               T" OR "OPIOID ANTAGONIST" PROSTAGLANDIN OR "ENDOTHELIN ANTAGONIST
               " OR "POTASSIUM CHANNEL ACTIVATOR"

=> s "calcium blocker" or alpha-blocker or beta-blocker or "ergot alkanoid" or
"vasoactive intestinal peptide" or "dopamine agonist" or "opioid antagonist" or
prostaglandin or "endothelin antagonist" or "potassium channel activator"
              0 "CALCIUM"
              0 "BLOCKER"
              0 "CALCIUM BLOCKER"
                  ("CALCIUM"(W)"BLOCKER")
              0 ALPHA
              0 BLOCKER
              0 ALPHA-BLOCKER
                  (ALPHA(W)BLOCKER)
              0 BETA
              0 BLOCKER
              0 BETA-BLOCKER
                  (BETA(W)BLOCKER)
              0 "ERGOT"
              0 "ALKANOID"
              0 "ERGOT ALKANOID"
                  ("ERGOT"(W)"ALKANOID")
              0 "VASOACTIVE"
              0 "INTESTINAL"
              1 "PEPTIDE"
              0 "VASOACTIVE INTESTINAL PEPTIDE"
                  ("VASOACTIVE"(W)"INTESTINAL"(W)"PEPTIDE")
              0 "DOPAMINE"
              0 "AGONIST"
              0 "DOPAMINE AGONIST"
                  ("DOPAMINE"(W)"AGONIST")
              0 "OPIOID"
              0 "ANTAGONIST"
              0 "OPIOID ANTAGONIST"
                  ("OPIOID"(W)"ANTAGONIST")
              0 PROSTAGLANDIN
              0 "ENDOTHELIN"
              0 "ANTAGONIST"
              0 "ENDOTHELIN ANTAGONIST"
                  ("ENDOTHELIN"(W)"ANTAGONIST")
              0 "POTASSIUM"
              0 "CHANNEL"
              0 "ACTIVATOR"
              0 "POTASSIUM CHANNEL ACTIVATOR"
                  ("POTASSIUM"(W)"CHANNEL"(W)"ACTIVATOR")
L3            0 "CALCIUM BLOCKER" OR ALPHA-BLOCKER OR BETA-BLOCKER OR "ERGOT
                ALKANOID" OR "VASOACTIVE INTESTINAL PEPTIDE" OR "DOPAMINE AGONIS
               T" OR "OPIOID ANTAGONIST" OR PROSTAGLANDIN OR "ENDOTHELIN ANTAGON
               IST" OR "POTASSIUM CHANNEL ACTIVATOR"

=> file biosis, medline, scisearch, embase
COST IN U.S. DOLLARS                          SINCE FILE      TOTAL
                                                 ENTRY    SESSION
FULL ESTIMATED COST                               1.08      18.75

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FILE 'SCISEARCH' ENTERED AT 17:11:21 ON 28 MAR 2007
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=> s "calcium blocker" or alpha-blocker or beta-blocker or "ergot alkanoid" or "vasoactive intestinal peptide" or "dopamine agonist" or "opioid antagonist" or prostaglandin or "endothelin antagonist" or "potassium channel activator"
L4 519080 "CALCIUM BLOCKER" OR ALPHA-BLOCKER OR BETA-BLOCKER OR "ERGOT ALKANOID" OR "VASOACTIVE INTESTINAL PEPTIDE" OR "DOPAMINE AGONIST" OR "OPIOID ANTAGONIST" OR PROSTAGLANDIN OR "ENDOTHELIN ANTAGONIST" OR "POTASSIUM CHANNEL ACTIVATOR"

=> s L4 and "chronic obstructive pulmonary"
L5 591 L4 AND "CHRONIC OBSTRUCTIVE PULMONARY"

=> display l5 all 591

L5 ANSWER 591 OF 591 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 75208458 EMBASE <<LOGINID::20070328>>
DN 1975208458
TI Personal experience with prostaglandin E2 in obstructive respiratory disease.
AU Maulide I.
CS Serv. Doencas Pulmon., Hosp. Sta Maria, Lisboa, Portugal
SO Pneumologia, (1974) Vol. 5, No. 4, pp. 241-247. .
CODEN: PNMLAB
DT Journal
FS 037 Drug Literature Index
003 Endocrinology
015 Chest Diseases, Thoracic Surgery and Tuberculosis
LA English
AB The main characteristics of the prostaglandins are described. The results are presented of a trial in which the responses to an aerosol of PGE2 were studied in 10 normal adults and 12 patients suffering from chronic obstructive pulmonary disease. A further study was carried out in 10 of the latter patients who are treated with PGE2 aerosol for 60 days. PGE2 has a bronchodilating action, particularly in non infected bronchioli. The mean duration of this effect is from 6 to 7 hr. Infection is an important factor to consider as it considerably reduces the effect of PGE2 in these patients. The difficulty in obtaining a metered aerosol of PGE2 constitutes a serious handicap for the ambulatory use of this substance in these patients.
CT Medical Descriptors:
*aerosol
*asthma
*bronchodilatation
*chronic obstructive lung disease
*clinical study
*breathing
theoretical study
major clinical study
therapy
inhalational drug administration
normal human
Drug Descriptors:
*prostaglandin
*prostaglandin e2
RN (prostaglandin e2) 363-24-6

=> s L5 and "phosphodiesterase inhibitor"
L6 13 L5 AND "PHOSPHODIESTERASE INHIBITOR"

=> display l6 all 13

L6 ANSWER 13 OF 13 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 1998154822 EMBASE <<LOGINID::20070328>>
TI New therapies for chronic obstructive pulmonary disease.
AU Barnes P.J.
CS Prof. P.J. Barnes, Department of Thoracic Medicine, National Heart and Lung Institute, Imperial College, London SW3 6LY, United Kingdom
SO Thorax, (1998) Vol. 53, No. 2, pp. 137-147. .
Refs: 139
ISSN: 0040-6376 CODEN: THORA7
CY United Kingdom
DT Journal; General Review
FS 006 Internal Medicine
015 Chest Diseases, Thoracic Surgery and Tuberculosis
030 Pharmacology
037 Drug Literature Index
LA English
ED Entered STN: 18 Jun 1998
Last Updated on STN: 18 Jun 1998
CT Medical Descriptors:
*chronic obstructive lung disease: DT, drug therapy
*chronic obstructive lung disease: TH, therapy
bronchodilatation
smoking cessation
beta adrenergic stimulation
immunomodulation
drug efficacy
drug mechanism
human
review
priority journal
Drug Descriptors:
*bronchodilating agent: DT, drug therapy
*bronchodilating agent: PD, pharmacology
*leukotriene b4 receptor antagonist: DT, drug therapy
*leukotriene b4 receptor antagonist: PD, pharmacology
*prostaglandin derivative: DT, drug therapy
*prostaglandin derivative: PD, pharmacology
*macrolide: DT, drug therapy
*macrolide: PD, pharmacology
*antioxidant: DT, drug therapy
*antioxidant: PD, pharmacology
*corticosteroid: DT, drug therapy
*corticosteroid: PD, pharmacology
misoprostol: DT, drug therapy
misoprostol: PD, pharmacology
butaprost: DT, drug therapy
butaprost: PD, pharmacology
erythromycin: DT, drug therapy
erythromycin: PD, pharmacology
clarithromycin: DT, drug therapy
clarithromycin: PD, pharmacology
roxithromycin: DT, drug therapy
roxithromycin: PD, pharmacology
1 [3 (cyclopentyloxy) 4 methoxyphenyl] 1 phenyl 2 (4 pyridyl)ethane: DT, drug therapy
1 [3 (cyclopentyloxy) 4 methoxyphenyl] 1 phenyl 2 (4 pyridyl)ethane: PD, pharmacology
immunomodulating agent: DT, drug therapy
immunomodulating agent: PD, pharmacology

muscarinic m2 receptor: EC, endogenous compound
darifenacin: DT, drug therapy
darifenacin: PD, pharmacology
muscarinic m3 receptor antagonist: DT, drug therapy
muscarinic m3 receptor antagonist: PD, pharmacology
revatropate: DT, drug therapy
revatropate: PD, pharmacology
atropine: DT, drug therapy
ipratropium bromide: DT, drug therapy
immunoglobulin enhancer binding protein: EC, endogenous compound
leukotriene b4: EC, endogenous compound
interleukin 8: EC, endogenous compound
2 [2 propyl 3 [3 [2 ethyl 4 (4 fluorophenyl) 5 hydroxyphenoxy]propoxy]phenoxy]benzoic acid: DT, drug therapy
2 [2 propyl 3 [3 [2 ethyl 4 (4 fluorophenyl) 5 hydroxyphenoxy]propoxy]phenoxy]benzoic acid: PD, pharmacology
7 [3 [2 cyclopropylmethyl 3 methoxy 4 [(methylamino)carbonyl]phenoxy]propoxy] 3,4 dihydro 8 propyl 2h 1 benzopyran 2 propanoic acid: DT, drug therapy
7 [3 [2 cyclopropylmethyl 3 methoxy 4 [(methylamino)carbonyl]phenoxy]propoxy] 3,4 dihydro 8 propyl 2h 1 benzopyran 2 propanoic acid: PD, pharmacology
1 [3 (4 biphenylylmethyl) 4 hydroxychroman 7 yl]cyclopentanecarboxylic acid: DT, drug therapy
1 [3 (4 biphenylylmethyl) 4 hydroxychroman 7 yl]cyclopentanecarboxylic acid: PD, pharmacology
sb 201146: DT, drug therapy
sb 201146: PD, pharmacology
phosphodiesterase inhibitor: DT, drug therapy
phosphodiesterase inhibitor: PD, pharmacology
4 cyano 4 [3 cyclopentyloxy 4 methoxy phenyl] 1 cyclohexanecarboxylic acid: DT, drug therapy
4 cyano 4 [3 cyclopentyloxy 4 methoxy phenyl] 1 cyclohexanecarboxylic acid: PD, pharmacology
cp 80633: DT, drug therapy
cp 80633: PD, pharmacology
unindexed drug
unclassified drug

RN (misoprostol) 59122-46-2, 59122-48-4; (butaprost) 69648-38-0; (erythromycin) 114-07-8, 70536-18-4; (clarithromycin) 81103-11-9; (roxithromycin) 80214-83-1; (darifenacin) 133099-04-4, 133099-07-7; (revatropate) 149926-91-0; (atropine) 51-55-8, 55-48-1; (ipratropium bromide) 22254-24-6; (leukotriene b4) 71160-24-2; (interleukin 8) 114308-91-7; (2 [2 propyl 3 [3 [2 ethyl 4 (4 fluorophenyl) 5 hydroxyphenoxy]propoxy]phenoxy]benzoic acid) 161172-51-6; (7 [3 [2 cyclopropylmethyl 3 methoxy 4 [(methylamino)carbonyl]phenoxy]propoxy] 3,4 dihydro 8 propyl 2h 1 benzopyran 2 propanoic acid) 141059-52-1, 153632-99-6, 153633-01-3, 153633-02-4; (1 [3 (4 biphenylylmethyl) 4 hydroxychroman 7 yl]cyclopentanecarboxylic acid) 158081-99-3

CN Ly 293111; Sc 53228; Cp 105696; Sb 201146; Sb 207499; Cp 80633; Cdp 840; Uk 88525; Uk 112166

=> s L5 and (zaprinast or dipyridamole or sildenafil)
L7 9 L5 AND (ZAPRINAST OR DIPYRIDAMOLE OR SILDENAFIL)

=> display l7 all 9

L7 ANSWER 9 OF 9 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 96123943 EMBASE <<LOGINID::20070328>>
DN 1996123943
TI Drug-induced lung disease: Cardiovascular agents.
AU Zitnik R.J.

CS Department of Internal Medicine, Yale University School of Medicine,New Haven, CT, United States
SO Journal of Respiratory Diseases, (1996) Vol. 17, No. 4, pp. 293-298. .
ISSN: 0194-259X CODEN: JRDIFQ
CY United States
DT Journal; General Review
FS 006 Internal Medicine
015 Chest Diseases, Thoracic Surgery and Tuberculosis
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
ED Entered STN: 7 May 1996
Last Updated on STN: 7 May 1996
AB The main respiratory side effect of angiotensin converting enzyme inhibitors is a nonproductive cough. Drug withdrawal is usually indicated; however, the cough may resolve spontaneously or respond to inhaled cromolyn. Although β -blockers are generally avoided in patients with asthma or chronic obstructive pulmonary disease (COPD), esmolol is the agent of choice when β- blockade is required for unstable angina. For patients with asthma or COPD who have had a myocardial infarction, low doses of atenolol or metoprolol may be given, but discontinue therapy at any sign of worsening respiratory function. Hydralazine can cause the development of a lupus-like syndrome, while protamine has been associated with anaphylaxis and pulmonary hypertension.
CT Medical Descriptors:
*angioneurotic edema: ET, etiology
*angioneurotic edema: DI, diagnosis
*angioneurotic edema: SI, side effect
*angioneurotic edema: TH, therapy
*coughing: DI, diagnosis
*coughing: SI, side effect
*coughing: TH, therapy
*coughing: ET, etiology
*drug induced disease: DI, diagnosis
*drug induced disease: DT, drug therapy
*drug induced disease: TH, therapy
*drug induced disease: SI, side effect
*drug induced disease: ET, etiology
*pulmonary hypertension: DT, drug therapy
*pulmonary hypertension: SI, side effect
*pulmonary hypertension: ET, etiology
*pulmonary hypertension: DI, diagnosis
anaphylaxis: SI, side effect
anaphylaxis: TH, therapy
anaphylaxis: DI, diagnosis
anaphylaxis: DT, drug therapy
anaphylaxis: ET, etiology
beta adrenergic receptor blocking
clinical feature
drug withdrawal
human
lung toxicity: DT, drug therapy
lung toxicity: TH, therapy
lung toxicity: SI, side effect
lung toxicity: ET, etiology
lung toxicity: DI, diagnosis
review
risk factor
systemic lupus erythematosus: ET, etiology
systemic lupus erythematosus: SI, side effect
systemic lupus erythematosus: TH, therapy
systemic lupus erythematosus: DT, drug therapy

systemic lupus erythematosus: DI, diagnosis
Drug Descriptors:
*cardiovascular agent: AE, adverse drug reaction
atenolol: AE, adverse drug reaction
beta adrenergic receptor blocking agent: AE, adverse drug reaction
calcium channel blocking agent
corticosteroid: DT, drug therapy
dipeptidyl carboxypeptidase inhibitor: AE, adverse drug reaction
dipyridamole: AE, adverse drug reaction
esmolol: AE, adverse drug reaction
histamine h1 receptor antagonist: DT, drug therapy
histamine h2 receptor antagonist: DT, drug therapy
hydralazine: AE, adverse drug reaction
isophane insulin: AE, adverse drug reaction
labetalol: AE, adverse drug reaction
metoprolol: AE, adverse drug reaction
pindolol: AE, adverse drug reaction
protamine: AE, adverse drug reaction
RN (atenolol) 29122-68-7; (dipyridamole) 58-32-2; (esmolol) 81147-92-4, 81161-17-3; (hydralazine) 304-20-1, 86-54-4; (isophane insulin) 9004-17-5; (labetalol) 32780-64-6, 36894-69-6; (metoprolol) 37350-58-6; (pindolol) 13523-86-9, 21870-06-4; (protamine) 11061-43-1, 9007-31-2, 9012-00-4
CN Neutral protamine hagedorn insulin

=> FIL STNGUIDE
COST IN U.S. DOLLARS SINCE FILE ENTRY 16.81 TOTAL SESSION 35.56

FILE 'STNGUIDE' ENTERED AT 17:14:43 ON 28 MAR 2007
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FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Mar 23, 2007 (20070323/UP).

=> file biosis, medline, scisearch, embase
COST IN U.S. DOLLARS SINCE FILE ENTRY 1.38 TOTAL SESSION 36.94

FILE 'BIOSIS' ENTERED AT 17:28:28 ON 28 MAR 2007
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FILE 'EMBASE' ENTERED AT 17:28:28 ON 28 MAR 2007
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=> s sildenafil and arginine
L8 591 SILDENAFIL AND ARGININE

=> s sildenafil and L-arginine
L9 382 SILDENAFIL AND L-ARGININE

=> s sildenafil (s) L-arginine
L10 178 SILDENAFIL (S) L-ARGININE

=> display l10 all 178

L10 ANSWER 178 OF 178 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

AN 1999313603 EMBASE <<LOGINID::20070328>>

TI The effect of sildenafil on apomorphine-evoked increases in intracavernous pressure in the awake rat.

AU Andersson K.-E.; Gemalmaz H.; Waldeck K.; Chapman T.N.; Tuttle J.B.; Steers W.D.

CS W.D. Steers, Department of Urology, Box 422, Univ. of Virginia Hlth. Sci. Center, Charlottesville, VA 22908, United States

SO Journal of Urology, (1999) Vol. 161, No. 5, pp. 1707-1712. .
Refs: 30
ISSN: 0022-5347 CODEN: JOURAA

CY United States

DT Journal; Article

FS 028 Urology and Nephrology
030 Pharmacology
037 Drug Literature Index

LA English

SL English

ED Entered STN: 27 Sep 1999
Last Updated on STN: 27 Sep 1999

AB Purpose: The aim of this study was to develop a quantitative, awake animal model to investigate the effect of sildenafil on centrally-evoked erectile activity. Methods: Intracavernous pressures were recorded in awake, male Sprague Dawley rats after administration of apomorphine (100 or 250 μg./kg. subcutaneously). Sildenafil (100 μg./kg. intravenously) was then given 10 min. after a second dose of apomorphine. The time to first response, duration of response, and peak intracavernous pressure and area under the response, were measured before and after sildenafil. Results: Apomorphine produced rhythmic increases in intracavernous pressure. The pressure increase consisted of two components. The amplitude of the first, tonic response was 58 ± 3 mm. Hg, and a superimposed, burst-like increase in pressure elevated this further to 81 ± 6 mm. Hg. Bilateral transection of the pudendal nerves abolished the burstlike pressure changes; bilateral transection of the cavernous nerves prevented both responses. The duration of the apomorphine- induced increase in intracavernous pressure was significantly ($p = 0.003$) prolonged by sildenafil (100 μg./kg.) from 37 ± 4 to 62 ± 11 s ($n = 6$). The overall intracavernous pressure response to apomorphine (100 μg./kg.), measured as the area under the curve, was significantly ($p = 0.003$) increased by sildenafil (100 μg./kg.) from 67 ± 8 to 142 ± 31 units ($n = 6$), N- nitro-L-arginine methyl ester (40 mg./kg. intravenously) prevented the apomorphine-induced responses. Conclusions: Monitoring intracavernous pressures in the awake rat represents a simple model to evaluate the effect of drugs on erectile function. Using this model we have shown that apomorphine elicits a rise in intracavernous pressure that can be prolonged by sildenafil. These results suggest that there may be a role for the combination of apomorphine and sildenafil in the management of erectile dysfunction.

CT Medical Descriptors:
*drug effect
*erectile dysfunction: DT, drug therapy
pressure measurement
area under the curve
impotence
pudendal nerve
nerve transection
drug potentiation
nonhuman
male
rat
animal experiment

animal model
controlled study
adolescent
intravenous drug administration
article
priority journal
Drug Descriptors:
*apomorphine: CB, drug combination
*apomorphine: IT, drug interaction
*apomorphine: DT, drug therapy
*sildenafil: CB, drug combination
*sildenafil: IT, drug interaction
*sildenafil: DT, drug therapy
RN (apomorphine) 314-19-2, 58-00-4; (sildenafil) 139755-83-2

=> s sildenafil and L-arginine
L11 382 SILDENAFIL AND L-ARGININE

=> display l11 382 all

L11 ANSWER 382 OF 382 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 1999114920 EMBASE <<LOGINID::20070328>>
TI Effects of castration and androgen replacement on erectile function in a rabbit model.
AU Traish A.M.; Park K.; Dhir V.; Kim N.N.; Moreland R.B.; Goldstein I.
CS Dr. A.M. Traish, Biochemistry and Urology, Department of Urology, Boston University School of Medicine, 700 Albany Street, Boston, MA 02118, United States. atraish@bu.edu
SO Endocrinology, (1999) Vol. 140, No. 4, pp. 1861-1868. .
Refs: 38
ISSN: 0013-7227 CODEN: ENDOAO
CY United States
DT Journal; Article
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AB We investigated, in a rabbit model, the effects of castration and testosterone replacement on: 1) the hemodynamics of the corpus cavernosum; 2) α-1 adrenergic receptor protein expression; 3) neural NO synthase protein expression and activity; 4) phosphodiesterase type 5 activity; and 5) trabecular smoothmuscle/connective tissue balance. One week after bilateral orchiectomy, animals were treated for 7 days with vehicle alone, testosterone, or estradiol. Intact control animals received vehicle only. Systemic arterial blood and intracavernosal pressures (ICP) were measured in each animal before and after electrical stimulation of the cavernosal nerve, α1-adrenergic receptor protein expression was determined by ligand binding studies. NO synthase expression and activity were determined by Western blot analyses and conversion of L-arginine to citrulline, respectively. Phosphodiesterase type 5 activity was determined by hydrolysis of guanosine 3',5'-cyclic monophosphate (cGMP) in tissue extracts in the absence or presence of 100 nM sildenafil. Smooth muscle content was assessed by Masson's trichrome staining and computer-assisted his tomorphometry. Castration significantly reduced ICP, but it did not alter systemic arterial blood pressure during stimulation of the cavernosal nerve. Testosterone, but not estradiol, treatment prevented the effects of castration and restored ICP to values similar to those obtained in intact animals. Castration reduced expression of α1-adrenergic receptor, and this reduction was prevented or reversed by restosterone replacement. Neural NO synthase protein expression and total activity were not altered significantly by

castration or after testosterone replacement. However, phosphodiesterase type 5 activity increased in castrated animals treated with testosterone. Castration significantly reduced trabecular smooth muscle content, and this reduction was restored by testosterone (but not estradiol) treatment. The results of this study demonstrate that androgen deprivation alters the functional responses and structure of erectile tissue.

CT Medical Descriptors:
*castration
*penis erection
rabbit
hormone substitution
corpus cavernosum
protein expression
structure activity relation
nonhuman
animal model
article
priority journal
Drug Descriptors:
*androgen
nitric oxide synthase

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